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Environmental Carbon Monoxide from Cigarette Smoking—A Critique

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It is often asserted that tobacco smoke in the atmosphere of public places constitutes a health hazard to exposed nonsmokers because of the environmental carbon monoxide generated during the burning of a cigarette. Cigarette smoking is an insignificant source of carbon monoxide in the overall atmosphere as compared with other natural and man-made sources. Even in tightly closed spaces with a large percentage of smokers, only rarely is it possible to build up concentrations which would exceed the established threshold limiting values for extended exposures. Examples include meeting rooms, houses, buses, and aircraft. Carboxyhemoglobin levels in nonsmokers resulting from carbon monoxide in environmental tobacco smoke are below the amount needed to produce the maximum allowable limit of 4% carboxyhemoglobin in the blood, as suggested by the World Health Organization. No strong evidence has been found indicating adverse effects in healthy individuals from concentrations of carboxyhemoglobin at or below these levels.

Recent publications of the Public Health Service (USDHEW) (42) have cited carbon monoxide (CO) as a harmful constituent in cigarette smoke. A number of investigators (43) have also suggested that smoking contributes significantly to the presence of environmental CO, especially in closed spaces, and thus constitutes a hazard to nonsmokers. The purpose of the present study was to explore this latter question and to define conditions to be encountered in various situations.

SOURCES OF CARBON MONOXIDE

It has been estimated that the earth's atmosphere at any given moment contains approximately 530 million metric tons of CO (35, 56) (Table 1). Natural and man-made sources produce annually 10 times this amount. Because of the continual consumption of CO in a number of oxidation reactions in the atmosphere and stratosphere, and in microbial actions in the soil, the half-life of atmospheric CO is only one-tenth of a year.

Cigarette smoking worldwide is estimated to produce only about 0.6 million tons of CO per year, as compared with the estimated total of over 5000 million tons from all sources. This estimate is based on an average figure of 0.100 g per cigarette for both mainstream and sidestream smokes. Clearly, if one is to be concerned about CO from smoking, it must be from a local concentration point of view, rather than from the total amount available in the atmosphere. For this reason, attention is usually focused on smoking in enclosed spaces such as rooms, buses, automobiles, airplanes, arenas, etc.

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TABLE 1
ANNUAL PRODUCTION AND LOSSES OF ATMOSPHERIC CARBON MONOXIDE (35, 56)

	Estimated production or loss (millions of tons)
Production	
Atmospheric oxidation of methane	5,000
Combustion of fossil fuels	270
Escape from oceanic solution	150
Tobacco smoking	0.6
Losses	
Atmospheric oxidation to CO_2	4,500
Stratospheric oxidation	500
Soil microorganisms	500
Equilibrium content	530

CONCENTRATIONS OF CO IN CLOSED SPACES

Carbon monoxide concentrations in nonurban outdoor atmospheres are usually less than 2 ppm. In highly populated cities, average concentrations may range as high as 10 ppm (13), depending on proximity to sources (highways or streets, factories, etc.) and air circulation (or stagnation). In general, outdoor concentrations are less than the ambient air quality standard of 9 ppm or the USA Environmental Protection Agency standard limit of 8 ppm for 24-hr exposure (15).

In enclosed spaces such as homes, offices, elevators, workshops, autos, buses, aircraft, etc., maximum equilibrium concentrations of CO buildup from smoking depend on rates of cigarette smoking, volume of spaces, ventilation rates, and CO losses (29). A large number of combinations have been investigated and measured (18, 21, 39, 44). Many of the tests were carried out under extreme conditions that are unrealistic in terms of usual human exposures.

Some recent results are listed in Table 2. It is immediately evident from these data that, under realistic conditions, CO concentrations from smoking are only one-fifth to one-tenth of those reported in sealed climatic chambers or unventilated rooms. Both Russell (44) and Harke *et al.* (20, 21) found that, in these closed, unventilated, smoke-filled rooms, people experience annoying eye and nose irritations long before the CO concentration in the room reaches 40 ppm.

The American Conference of Governmental Industrial Hygienists (53) has established a threshold limiting value for CO of 50 ppm as the maximum for daily 8-hr exposure. Jones and Fagan (29) have shown that in a closed room or house it is almost impossible to attain this concentration of CO by smoking alone. The reason for this is that, unless the space is tightly sealed, "normal" leakage through cracks around doors and windows permits substantial circulation of air into and out of the room (26), usually sufficient to change the total volume of air at least once every hour. There seems to be a practical, upper limit to the possible buildup of CO concentration from smoking in the real-life situation.

In this connection, the modern jet passenger aircraft is probably the most effectively ventilated enclosed space occupied by humans, since a complete change of air occurs every 3 min. Even if all the 165 passengers in a full Boeing 707 aircraft

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TABLE 2
MAXIMUM CO CONCENTRATIONS RECENTLY REPORTED IN ENCLOSED SPACES

Situation	Estimated smoke generation rate (cigs/m³/hr)	Ventilation (air changes/hr)	Maximum CO (ppm)
With little or no ventilation			
Room 58 m³ (19)	2.6	None (?)	50
Room 43 m³ (44)	1.4	None (?)	38
Small autos on Hamburg streets (23)	~3.0	Vents closed	24.3
Stationary auto (22)	~3.0	Vents closed	110.4
Unventilated climate chamber 30 m³ (28)	6.0	0.1	85 at 10 min
With normal ventilation			
Room 58 m³(19)	2.6	8.8	10
Party of 50 people in 170-m³ room (9)	0.26	7.0	7
Room 80 m³ (1)	0.3	6.4	6
Small autos on Hamburg streets (23)	~3.0	Vents open	12.0
Stationary auto in wind tunnel (22)	~3	~60	5-6

were smoking at the rate of four cigarettes per hour, the ambient CO concentration would only increase by 12 ppm. The levels measured in actual flights averaged 2–3 ppm (range 0–5 ppm) (25).

These examples serve to emphasize the importance of reasonable ventilation as a means of reducing interior environmental pollution of any kind. Spaces which are ventilated to minimize offensive odors and to provide comfort conditioning are

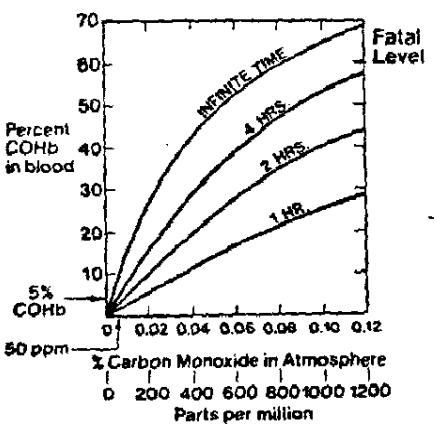

FIG. 1. Effects of high levels of carbon monoxide (16). Note that in the lower left-hand corner are shown the value of 5% COHb, the level found for the average smoker in an urban environment, and the atmospheric concentration value of 50 ppm, the threshold limiting value established by the American Conference of Government Industrial Hygienists (53).

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usually also free of excessive concentrations of carbon monoxide and other potentially annoying or irritating gases or aerosols, even though cigarette smoking is taking place (47). In such situations it should not be necessary to designate smoking areas or to impose prohibition.

INHALED CO AND CARBOXYHEMOGLOBIN

The main effect (if not the only effect) of inhaling CO is the formation of carboxyhemoglobin (COHb) in the blood (Fig. 1). CO binds strongly to hemoglobin, preventing it from performing its intended function of transporting oxygen to the tissues and carbon dioxide to the lungs. The level of COHb (expressed as a percentage of the hemoglobin so taken up) depends upon the concentration of CO in the lungs, the time of exposure, the rates of respiration and heart beat, exercise factors, etc. (24, 27, 30, 32, 40). Eventually, under constant conditions, an equilibrium concentration of COHb is established (11, 31). The formation of COHb is reversible, so that, at lower concentrations of CO, the excess COHb breaks down to release CO and free hemoglobin. Obviously, at high enough levels of COHb (50–70%), oxygen needed by various tissues is no longer supplied in adequate amounts and various adverse effects on body functions are observable. That such effects do not take place from inhaling environmental cigarette smoke is shown later.

Under the relationships between environmental CO from smoke and COHb, the only important question is: How much COHb is formed in the exposed subjects? In fact, this is the only complete statement one can make about CO exposure. Statements limited to transient concentrations of CO in smoke or in the atmosphere are inadequate to define what is taking place. Inferences from such information about the effects of CO are usually based on unrealistic assumptions concerning the other conditions involved. For this reason, most of the information in the literature about CO concentrations from smoking is not meaningful in terms of human experience.

OBSERVED COHb CONCENTRATIONS

The literature contains many studies on the COHb concentrations in smokers and in nonsmokers under various circumstances. Such investigations are invariably complicated by the fact that differing CO concentrations are found in various environments, even in the absence of smoking. In fact, the human body itself generates CO *de novo*, so that the blood always contains from 0.4 to 1% COHb (18, 41). Table 3 summarizes some of the recent findings.

The pattern from these data is very clear. Nonsmokers will generally have less than 2% COHb, depending upon the situation to which they are exposed. Only in extreme situations, such as those used by Russell *et al.* (44) in their experiments, will nonsmokers experience COHb levels in excess of 2% from cigarette smoke-filled environments. In their experiment, 21 persons were crowded into a sealed chamber, 15 × 12 × 8 ft, and exposed to the smoke of 80 cigarettes and two cigars in a 78-min period. The CO concentration rose to a maximum of 38 ppm, and the average COHb for the nonsmokers increased from 1.6 to 2.6%. In the description of Russell *et al.* (44): "The exposure was very unpleasant, causing eyes to burn and water. For most volunteers it was worse than they could recall having tolerated on normal occasions."

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TABLE 3
CARBOXYHEMOGLOBIN LEVELS IN SMOKERS AND NONSMOKERS

Description	Mean (m) or range (r) of % COHb	
	Nonsmokers	Smokers
UK pregnant women (24)	(m) 1.1	(m) 3.6
Meat porters (24)	(m) 1.6	(m) 5.1
Office workers (24)	(m) 1.3	(m) 6.2
London office workers (10)	(m) 1.12 (r) 0.1–2.7	(m) 5.5 (r) 2.2–13.0
29,000 USA blood donors (49)	(m) 1.39 (r) 0.4–6.9	(m) 5.57 (r) 0.8–11.9
3311 California longshoremen (17)	(m) 1.3	(m) 5.9
Munich population (46)	(m) 2.36	(m) 7.38
Rural Bavarians (46)	(m) 1.03	(m) 6.06

The World Health Organization (57) has suggested a maximum of 4% COHb as a level which is safe from all possible adverse effects under continuous exposure conditions. In order to reach this level, the nonsmokers in the situation Russell *et al.* designed would have had to remain in the intolerably smoky room for at least another 78 min.

Harke *et al.* (21) carried out similar experiments and made the following concluding statement:

"It does not appear possible that such smoke densities can be generated by smokers. This experiment using smokers (rather than smoking machines) would only be reproducible when

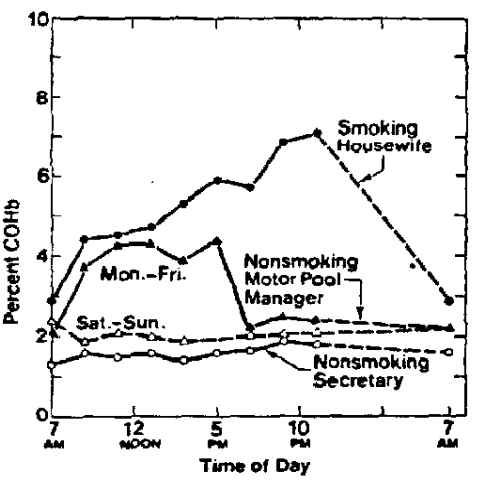

FIG. 2. Temporal variations of COHb concentration (37). Note that the COHb level of the nonsmoking motor pool manager increases during the day when he is at work and quickly drops again at the end of the work day. The smoking housewife would have a daily average of about 5%, characteristic of smokers (the value shown in the lower left-hand corner of Fig. 1).

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50 persons were placed in the room (floor space 62 m³), and each of them would smoke three cigarettes during a period of 30 minutes. The irritation . . . would be so great that certainly a large majority of the smokers would have to leave the room prior to the end of the smoking period."

It should be emphasized that, because of the dynamic interchange of CO between the inhaled air and the COHb in the blood, the level of COHb is constantly changing, depending on circumstances. McIlvaine and associates (37) have measured COHb values at 2-hr intervals throughout the day for smokers and nonsmokers. Some results are shown in Fig. 2. Obviously, the definition of an average condition of exposure to CO presents some difficulties.

RESPONSE OF HEALTHY INDIVIDUALS TO INCREASED COHb

Many investigators have carried out performance and pathological tests aimed at showing that increased COHb in the blood impairs the well-being of the persons exposed to CO in the environment (8, 14, 36, 55). Performance tests are generally directed at demonstrating the impairment of oxygen-sensitive organs such as the eye and the brain. These researchers indicate that no discernible effects occur in normal healthly adults below 10% COHb. Some effects may be observed at relatively high and unusual COHb levels (15% and over).

Recently, a number of studies have been carried out to explore the hypothesis that CO exposure contributes to pathological changes leading to atherosclerosis (54). Pigeons (3), rabbits (7, 51, 52), dogs (7), primates (7, 12, 33, 50), and man (34, 38) have all been investigated. No firm indications of detrimental effects have so far been found at COHb levels of less than 15%.

The recommendation by WHO of 4% COHb as the maximum safe level is considered by this reviewer as cautiously conservative. The evidence to date indicates that only if the individual is handicapped with severe heart disease might he have some difficulties at the 4% COHb level (5, 6), and even then the probability is very low. Such a person may lack sufficient reserve to compensate for such loss in oxygen carrying capacity of the blood. Hence, if he were also exercising for

TABLE 4

HUMAN RESPONSE TO ELEVATED CARBOXYHEMOGLOBIN IN THE BLOOD (48)

% COHb	Healthy adult	Heart patients
0.3–0.7	Normal	Normal
1–5	Increased blood flow	May feel lack of oxygen
2–9	Exercise tolerance reduced	Chest pains with less exertion
16–20	Headache, lowered visual response	May be lethal for patients with severe cardiac problems
20–30	Throbbing headache, nausea, impaired manual dexterity	
30–40	Severe headache, nausea, and vomiting	
50	Coma, convulsions	
67–70	Lethal	

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a considerable period of time, he might experience early onset of chest pains.

Stewart (48), in his overall review of the physiological effects of CO, has published the information shown in Table 4 summarizing human responses to various levels of COHb.

CONCLUSIONS

The following conclusion, which appears in the workshop report entitled "Environmental Tobacco Smoke Effects on the Non-Smoker," published in the Scandinavian Journal of Respiratory Diseases (45), is supported by many investigators who have studied this problem (2, 4).

> "Concentrations that are present under realistic environmental conditions may reach about 10 ppm. It is true that higher concentrations have been reported, but they represent only transient values or levels reached under experimental conditions. If the exposure to 10 ppm were to prevail for 8 hours the resulting COHb concentration would be 1.9%. This value is well below the suggested WHO maximum of 4%. At these levels no adverse biological effects will occur in a population that can be expected to be present in such environments. It can thus be concluded that the carbon monoxide in environmental tobacco smoke does not represent a health hazard."

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